AMENDMENTS TO THE CLAIMS

1. (Canceled)

2. (Currently amended) ~~Use~~ Method according to claim 17 wherein the antagonist of the CB1 receptor is a specific antagonist of theCB1 receptor.

3 - 11. (Canceled)

12. (Currently amended) ~~Use~~ Method according to any of the preceding claims claim 17 wherein the CB1 receptor is selected from the group consisting of:

a) a protein having an amino acid sequence comprising SEQ ID NO: 1 or a portion of SEQ ID NO :1, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

b) a protein having an amino acid sequence comprising SEQ ID NO : 2 or a portion of SEQ ID NO : 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

c) an allele of the protein having the amino acid sequence of SEQ ID NO :1 or SEQ ID NO : 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

d) a protein having the amino acid sequence of SEQ ID NO :1 with a Phenylalanine to Leucine substitution at position 200; and/or an Isoleucine to Valine substitution at position 216; and/or a Valine to Alanine substitution at position 246;

e) a protein having the amino acid sequence of SEQ ID NO : 2 with a Phenylalanine to Leucine substitution at position 139; and/or an Isoleucine to Valine substitution at position 155;and/or a Valine to Alanine substitution at position 185; and

f) a protein comprising the amino acid sequences of SEQ ID NO : 3, SEQ ID NO : 4, SEQ ID NO :5, SEQ ID NO : 6, SEQ ID NO : 7, SEQ ID NO : 8 and SEQ ID NO : 9 or amino acid sequences 80% homologous to these, said protein having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

13. (Currently amended) ~~Use~~ Method according to ~~claims 1 to 11~~ claim 17 wherein the CB1

receptor is a protein having a homology at the amino acid level with SEQ ID NO : 1 of at least 45%, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

14. (Currently amended) ~~Use~~ Method according to the preceding claim 13 wherein the homology is at least 60%~~, preferably 70 %, more preferably 80 %, even more preferably 90 % and more preferably 95 %~~.

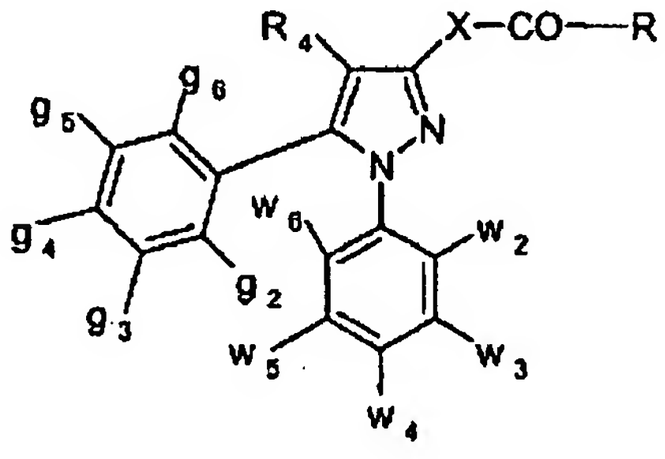
15. Currently amended) ~~Use~~ Method according to any of the preceding claims claim 17 wherein the daily dosage of CB1 receptor antagonist is from 0.01mg to 500mg~~, preferably from 1 mg to 100 mg~~.

16. (Canceled)

17. (Original) A method of treatment of hepatic diseases in a mammal comprising the administration of a therapeutically effective amount of at least one CB1 receptor antagonist to a mammal in need thereof.

18. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist is a compound of the formula II or one of its pharmaceutically acceptable salt, in which g_2, g_3, g_4, g_5 and g_6 and w_2,w_3, w_4, w_5 and w_6 are identical or different and are independently hydrogen, a chlorine or bromine atom, a (C_1-C_3) alkyl, a (C_1-C_3) alkoxy, a trifluoromethyl or a nitro group and g_4 is optionally a phenyl group; R_4 is hydrogen or a (C1-C3) alkyl ; X is either a direct bond or a group -$(CH_2)_x$-$N(R_3)$-, in which R_3 is hydrogen or a (C_1-C_3) alkyl and x is zero or one; R is: a group –NR_1R_2 in which R_1 and R2 are independently a (C_1-C_6)-alkyl; an non-aromatic (C_3-C_{15}) carbocyclic radical which is optionally substituted, said substituent (s) being other than a substituted carbonyl; an amino(C_1-C_4) alkyl group in which the amino is optionally disubstituted by a (C_1-C_3) alkyl ; a cycloalkyl (C_1-C_3) alkyl in which the cycloalkyl is C_3-C_{12} ; a phenyl which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C_1-C_5) alkyl or by a (C_1-C) alkoxy; a phenyl (C_1-C_3) alkyl ; a diphenyl (C_1-C_3) alkyl ; a naphthyl; an anthracenyl; a saturated 5-to 8-membered heterocyclic radical which is

unsubstituted or substituted by a (C_1-C_3) alkyl, by a hydroxyl or by a benzyl; a 1-adamantylmethyl; an aromatic heterocycle which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C_1-C_5) alkyl or by a (C_1-C_5) alkoxy; a (C_1-C_3) alkyl which is substituted by an aromatic heterocycle which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C_1-C_5) alkyl or by a (C_1-C_5) alkoxy; or else R, is hydrogen and R2 is as defined above; or else Rl and R2 form a saturated 5-to 8-membered heterocyclic radical with the nitrogen atom to which they are bonded, said heterocyclic radical being other than morpholine when w_2, w_3, w_4, w_5, w_6, g_2, g_3, g_4, g_5 and g_6 are all hydrogen; a group R_2 as defined above when X is -$(CH_2)_x$ $N(R_3)$- ; a group R_5 when X is a direct bond, R_5 being a (C_1-C_3) alkyl ; a (C_3-C_{12}) cycloalkyl which is unsubstituted or substituted by a (C_1-C_5) alkyl ; a phenyl(C_1-C_3) alkyl which is unsubstituted or substituted by a halogen or by a (C_1-C_5) alkyl; a cycloalkyl (C_1-C_3) alkyl in which the cycloalkyl is C_3-C_{12} and is unsubstituted or substituted by a(Cl-C5) alkyl ; or a 2-norbornylmethyl



(II)

19. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist is N-piperidono-3-pyrazolecarboxamide or one of its pharmaceutically acceptable salt.

20. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist isN-piperidino-5- (4-bromophenyl)-1- (2, 4-dichlorophenyl) -4-

ethylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

21. (Original) A method of treatment of hepatic diseases according to claim 17 wherein the CB1 receptor antagonist isN-piperidino-5- (4-chlorophenyl)-1- (2, 4-dichlorophenyl) -4-methylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

22. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~ claim 17 wherein the hepatic disease is liver fibrosis.

23. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~ claim 17 wherein the hepatic disease is alcoholic liver cirrhosis.

24. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~ claim 17 wherein the hepatic disease is chronic viral hepatitis.

25. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~ claim 17 wherein the hepatic disease is non-alcoholic steatohepatitis.

26. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~ claim 17 wherein the hepatic disease is primary liver cancer.

27. (Currently amended) A method of treatment of hepatic diseases according to ~~claims 17 to 21~~ claim 17 wherein the daily dosage of CB1 receptor antagonist is from 0.01mg to 500mg~~, preferably from 1mg to 100 mg~~.